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TITLE: Comparison of short-term energy intake and appetite responses to active and seated video gaming, in 8 to 11 year-old boys.

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RUNNING TITLE: Video gaming, energy intake and appetite.

KEY WORDS: Video gaming: Energy intake: Appetite: Satiety: Children.

28 **ABSTRACT**

29 The acute effects of active and seated video gaming on energy intake (EI), blood glucose, plasma
30 glucagon-like peptide-1 (GLP-1₇₋₃₆) and subjective appetite (hunger, prospective food consumption
31 and fullness) were examined in 8-11 y boys. In a randomised, crossover fashion, 22 boys completed
32 one, 90 min active and one, 90 min seated video gaming trial during which food and drinks were
33 provided *ad-libitum*. Energy intake, plasma GLP-1₇₋₃₆, blood glucose, and subjective appetite were
34 measured during and following both trials. Time averaged AUC blood glucose was increased
35 ($p=0.037$) however, EI was lower during active video gaming (1.63 ± 0.26 MJ) compared to seated
36 video gaming (2.65 ± 0.32 MJ) ($p=0.000$). In a post-gaming test meal 1 h later, there were no
37 significant differences in EI between the active and seated gaming trials. Although estimated energy
38 expenditure (EE) was significantly higher during active video gaming, there was still no
39 compensation for the lower EI. At cessation of the trials, relative energy intake (REI) was
40 significantly lower following active video gaming (2.06 ± 0.30 MJ) versus seated video gaming
41 (3.34 ± 0.35 MJ) ($p=0.000$). No significant differences were detected in time averaged AUC GLP-1₇₋
42 ₃₆ or subjective appetite. At cessation of the active video gaming trial, EI and relative EI was
43 significantly less than for seated video gaming. In spite of this, the relative EI established for active
44 video gaming was a considerable amount when considering the total daily EAR for 8-11 y boys in
45 the UK (7.70 MJ).

46 INTRODUCTION

47 In England, one sixth of children aged between 2-10 y are estimated to be obese ⁽¹⁾. Peak incidence
48 of obesity appears to be during mid-to-late childhood, when aged between 7 and 11 y, particularly
49 in boys ⁽²⁾. Physical activity (PA) in childhood is key as it lowers the risk of obesity and the related
50 chronic and life-limiting conditions such as cardiovascular disease and type 2 diabetes ⁽³⁾. The most
51 recent data for England indicates, that only 18.5% of children are achieving at least 60 min of
52 moderate to vigorous physical activity (MVPA) per day ⁽¹⁾. Moreover there is evidence of a decline
53 in PA as children progress into adolescence ^(1; 4). One reason for this decline in PA could be a
54 greater use of sedentary screen based media ⁽⁵⁾.

55 Active video gaming could however, provide a suitable replacement to seated based video game
56 play to potentially increase children's PA. Active video games integrate body movement (isolated
57 limbs or whole body) into the game experience and video gaming. Movements are sensed via a
58 hand-held motion controller (Nintendo Wii™), video cameras (Sony, Eye Toy™ and Microsoft,
59 Xbox Kinect™) or weight-sensing platforms (Konami, Dance Dance Revolution™ and Nintendo
60 Wii Fit™) ⁽⁶⁾. Recent active video gaming research with paediatric groups has established that game
61 play produces greater EE and light to moderate PA when compared with resting, television viewing
62 and seated video gaming ^(7; 8; 9; 10; 11; 12). There is also evidence that boys expend more energy than
63 girls during active video game play ^(8; 13) and that they display greater enjoyment and engagement in
64 this type of video game ⁽¹³⁾. In view of the aforementioned findings therefore, active video game
65 play might be more of a feasible alternative to seated video gaming for boys to increase PA levels.

66 In paediatric groups however, it appears that spontaneous EI both during and following sedentary
67 screen-based media activity ^(14; 15) occurs, which exceeds the energy expended and this could also
68 occur during active video gaming. Recent active video gaming research has observed that EI can
69 exceed EE following 1 h of game play in both 13-17 y males ⁽¹⁶⁾ and 12-15 y obese males ⁽¹⁷⁾.
70 Furthermore, when the 13-17 y males were monitored over 24 h after the trial, there was also a
71 down-regulation in PA following active video gaming. Consequently, similar to matched seated
72 video gaming and resting trials, the 13-17 y males were found to be in a positive energy balance
73 state following active video gaming ⁽¹⁶⁾.

74 Energy intake following active video game play ^(16; 18) however, might not be representative of
75 children's real-life active video gaming practices particularly as there is evidence of eating during
76 play ^(19; 20). Recently and in view of this, the EI from food and drinks offered *ad-libitum* during both

active and seated video gaming was explored, in 8-11 y boys⁽¹¹⁾. The findings of the study established that EE was significantly greater from active video game play, however EI during both trials was similar. As a result, the energy expended by active video game play, did not counterbalance the EI during it⁽²¹⁾ yet despite this, relative energy intake (REI) at cessation of gaming was significantly lowered. In the cited study, PAEE and EI were not monitored beyond the gaming trials so it is unknown whether any compensation occurred at a later time-point. In addition, subjective appetite sensations (hunger, fullness and prospective food consumption) which were similar during both active and seated video gaming, provided no explanation for the similarity in EI⁽¹¹⁾. Energy intake both during and following active and seated video gaming should therefore be measured. In addition, appetite should be measured objectively as well as subjectively, to explore whether homeostatic-related signals can provide an explanation for the EI during both active and seated video game play. Only one seated video gaming study thus far has measured appetite signals related to hunger, alongside subjective appetite in 15-19 y males⁽¹⁴⁾. No differences were found in total ghrelin or serum insulin between trials during seated video gaming, yet blood glucose was significantly higher. According to the 'glucostatic theory' of short-term appetite regulation a rise in glucose is indicative of a satiety response⁽²²⁾, yet the test meal EI of the 15-19 y males following the 1 h seated gaming bout was greater and post-gaming hunger sensations were not increased⁽¹⁴⁾. Satiety-related homeostatic signals were not measured in this earlier study⁽¹⁴⁾ so it is unknown whether appetite signals related to satiety were raised. The measurement of satiety-related appetite might have provided an explanation for the increased EI following seated gaming or given an indication as to whether it may be due to hedonic mechanisms^(14; 23; 24).

Due to the lack of difference in hunger-related signals during seated video gaming in 15-19 y males⁽¹⁴⁾ and the similarities in EI and appetite sensations of 8-11 y boys both during active and seated video gaming, it would be pertinent to measure satiety-related appetite signals. The measurement of satiety-related signals alongside subjective appetite could provide a more in-depth understanding of the mechanisms behind the spontaneous EI observed during both active and seated video gaming. The present study therefore, aimed to assess acute EI, plasma GLP-1₇₋₃₆, blood glucose and subjective appetite responses during and 1 h following 90 min bouts of active and seated video gaming, in 8-11 y boys.

MATERIALS AND METHODS

Design

108 A randomised, crossover design was used to compare plasma GLP-1₇₋₃₆, blood glucose, subjective
109 appetite and EI responses of 8-11 y-old boys, to active video gaming versus seated video gaming,
110 each separated by 1 week. The active video gaming bouts utilised were representative of children's
111 real-life active video gaming practices i.e. the type of active video game and console, the duration
112 (min) and EI during gaming were identified in a previous study ⁽¹⁹⁾. There were two gaming bouts:
113 (1) 90 min of seated video gaming and (2) 90 min of active video gaming. During each gaming
114 bout, food and drinks were offered *ad-libitum* enabling EI to be measured whilst gaming and also in
115 a test meal 1 h later. The boys were placed into groups of two according to school year. Each group
116 was then randomly assigned to a different bout every week so that by the end of the 2 weeks they
117 had completed each trial.

118 The study was conducted according to 2013 Declaration of Helsinki (World Medical Association.
119 2013) ⁽²⁵⁾ and was approved by the University of Northumbria, Faculty of Health and Life Sciences
120 Ethics Committee. Written informed consent was obtained from each child's parent and assent from
121 every boy, prior to data collection.

122

123 **Participants**

124 To recruit 8-11 y boys, consent was obtained from the head teacher of a Primary School located
125 within the city of Newcastle upon Tyne (North East England, UK). Recruitment packs were
126 distributed to all eligible boys who expressed an interest in participating and they were asked to take
127 it home to their parents. The pack contained a letter addressed to their parents with a full
128 explanation of the study and consent forms for them and their child (if able) to sign and return to
129 school. Signed consent forms were received from 22 boys (mean age 9.9±0.2 y). Boys were
130 excluded if they had intolerances or allergies to the foods provided in the study or had an injury or
131 illness which prevented their play of active video games. Overall, 22 boys participated in the study.

132 **Preliminary measures**

133 Prior to the first gaming trial, the researchers met the children (and where applicable, their parent) at
134 the school for familiarisation. The boys were familiarised with the gaming consoles (Nintendo
135 Wii™), games (Nintendo Wii™ Sports, tennis) (Nintendo ©), the gaming session format, the self-
136 reported weighed food diaries and visual analogue scales (VAS) that were used to explore
137 subjective appetite sensations. The researchers demonstrated the right hip placement of
138 accelerometers (Actigraph LLC © GT3X+) used for the measurement of PA during the gaming

139 trials. The boys were asked to complete a food preference questionnaire to ensure they did not
140 dislike the foods and drinks offered during the study. They completed the Dutch Eating Behaviour
141 Questionnaire for children (DEBQ-C), as a measure of dietary restraint ⁽²⁶⁾. Stature and seated
142 height were measured to the nearest 0.01 m using a Harpenden Portable Stadiometer (Holtain
143 Limited, Pembs, UK). Body mass was measured to the nearest 0.1 kg using portable SECA scales
144 (SECA United Kingdom). Waist circumference was measured to the nearest 0.01 m with a non-
145 elastic flexible tape at each boy's natural waist whilst standing as an indication of central adiposity
146 ⁽²⁷⁾.

147 **Protocol**

148 Each boy was provided with a self-report, weighed food diary and a set of food weighing scales
149 (Salter [®], Kent, UK). With the help of their parent they were asked to weigh and record all foods
150 and drinks they consumed from 1700 h the evening before, until after they had consumed breakfast
151 on the morning of each trial day. A photocopy of the food diary was provided to each parent who
152 was asked to replicate their child's food and drink intake prior to the second gaming trial. With the
153 help of school staff and parents, the boys abstained from all physical education at school on the day
154 of the study and PA from 1700 h the preceding evening.

155 On the trial days, the boys were met at school at 0830 h by two members of the research team and
156 escorted to the University laboratory. On arrival (~ 0850 h), the boys rested until 0900 h when they
157 completed baseline appetite VAS. Immediately following this (t=0 min), a finger-prick blood
158 sample (300 µL) was taken from each boy to enable the determination of baseline plasma GLP-1₇₋₃₆
159 and blood glucose.

160 The boys completed additional appetite VAS at 45 min during gaming, at the end of gaming (90
161 min), 45 min post gaming (135 min) and immediately following the test meal (180 min). Further
162 fingertip blood samples (300 µL) were taken at 45 min during gaming, at the end of gaming (90
163 min), 45 min post gaming (135 min) for the determination of plasma GLP-1₇₋₃₆ and blood glucose.
164 Upon termination of each 90 min gaming trial, the boys rested for 60 min following which they
165 were provided with an *ad-libitum* test meal, before being returned to school by the research team. A
166 diagrammatic representation of the study protocol is provided in Figure 1.

167 ***Gaming trials***

168 The design of the individual gaming trials was based on published data which described the active
169 gaming practices of 7-11 y children from Newcastle upon Tyne ⁽¹⁹⁾. The active video gaming
170 console utilised was Nintendo Wii™ and the game was Nintendo Wii™ Sports tennis ⁽¹⁹⁾. The
171 seated video game utilised was ‘Mario and Sonic at the London 2012 Olympic Games’ which was
172 played on the handheld device, Nintendo® 3DS. The two gaming trials took place on the same
173 school day of each week over two consecutive weeks. The two gaming trials were: 1) 90 min seated
174 video gaming during which food and drinks offered *ad-libitum*; 2) 90 min active video gaming
175 during which food and drink offered *ad-libitum* as they have been successfully used in previous
176 gaming and appetite work with young boys ⁽²¹⁾.

177 ***Energy intake***

178 The food and drink items provided during the gaming sessions were based on previous findings
179 ⁽¹⁹⁾ and comprised 130 g apples (raw, slices and cored), 50 g crisps [potato chips (Walkers®, ready
180 salted)], 250 mL semi-skimmed milk, and 250 mL ‘Jucee’ apple and blackcurrant squash (no added
181 sugar). All food items were pre-weighed by the researchers to the nearest gram using electronic
182 portable scales (Salter®, Kent, UK) and all drinks were measured to the nearest millilitre. The
183 crisps and apple were placed in clear plastic bags and the milk and squash were placed in coloured
184 drinks bottles so that volumes could not be detected. All items were numerically coded by the
185 researchers and placed at a station designated to each individual boy who were offered them *ad-*
186 *libitum*. When the gaming trials commenced, the time of the first eating episode for each boy was
187 recorded. The researchers noted each bag or bottle taken by the boys and anything left over was
188 weighed or measured so that amounts consumed could be calculated and recorded. Food and drink
189 items were topped up before being finished, during the gaming bouts.

190 The *ad-libitum* test meal provided after the gaming bouts was pasta, with tomato sauce, cheddar
191 cheese and olive oil (ASDA, Leeds, UK) which was served in excess and topped up before being
192 finished. The boys were instructed to eat until they felt comfortably full, at which point the meal
193 was terminated. As they ate the test meal, the bowl was refilled by the researchers. The research
194 team covertly weighed the test meal before it was served and as the meal was terminated. The
195 macronutrient content of the meal was 58% CHO, 28% fat and 14% protein and provided 450 kJ
196 (107.5 kcal) per 100 g of total energy, similar to a pasta meal utilised in a previous adolescent
197 appetite study ⁽²⁸⁾. Energy intake for all of the food and drink items served was estimated from
198 individual food labels, an online resource (www.asda.com) and MicroDiet (Downlee Systems®,
199 Derbyshire, UK).

200 ***Physical activity assessment***

201 During both gaming bouts, the PA levels of each boy were measured by accelerometry using an
202 Actigraph[®] LLC, GT3X+ placed on the right hip since this is considered the optimum site for PA
203 monitoring⁽²⁹⁾. Physical activity counts were recorded in 10 s epochs. Following each trial, the
204 accelerometer data was downloaded utilising Actilife 6 data analysis software and interpreted using
205 recommended child-appropriate activity cut-off values⁽³⁰⁾. Activity counts were converted into
206 mean metabolic equivalents (METs) using MET thresholds recommended for use with children:
207 sedentary < 1.5 METs; light 1.5 to < 4 METs; moderate 4 to < 6 METs; vigorous > 6 METs⁽³¹⁾.

208 ***Energy expenditure***

209 For each boy, Henry's body mass, stature and sex-specific equations were used to calculate basal
210 metabolic rate (BMR)⁽³²⁾. Energy expenditure was then calculated as recommended by Ridley,
211 Ainsworth and Olds⁽³³⁾, as follows; METs x BMR (MJ·min⁻¹·d) x 90 min gaming = MJ.

212 ***Relative energy intake***

213 For each boy, EE was subtracted from the amount of energy consumed during each 90 min gaming
214 bout and also from the test meal to calculate REI.

215 ***Subjective appetite***

216 Hunger, fullness and prospective food consumption were assessed using VAS. Questions asked
217 were: 'How hungry do you feel now?' accompanied by the statements, very hungry (0) and not at
218 all hungry (100); 'How full do you feel now?' accompanied by, very full (0) and not full at all
219 (100), and prospective food consumption "How much would you like to eat now?" accompanied
220 by a lot (0) and nothing at all (100). The boys were requested to place a vertical mark along the 100
221 mm horizontal lines. Scales were collected prior to the commencement of gaming (baseline t=0
222 min), at 45 min during gaming, at the end of gaming (90 min), 45 min post gaming (135 min) and
223 immediately following the test meal (180 min).

224 ***Blood sampling***

225 To obtain blood samples, the fingertip capillary blood sampling and handling method utilised by
226 Green and colleagues⁽³⁴⁾ was followed. For the measurement of GLP-1₇₋₃₆, capillary blood samples
227 were collected immediately before the gaming bouts commenced (baseline t=0 min), midway
228 during the gaming bout (45 min), at the end of the gaming bout (90 min) and at 45 min post-gaming
229 (135 min) (Figure 1). The fingertip puncture site was cleaned with an aseptic wipe then pierced with
230 a sterile automated lancet (Accu-Check, Mannheim, Germany). The blood was collected into a pre-

231 cooled EDTA microvette pre-treated with aprotinin (33 μ L per mL) and DPP-IV inhibitor (30 μ L
232 per mL) to aid in the preservation of GLP-1₇₋₃₆. Immediately following blood collection, the
233 microvettes were placed on ice and spun at 1500 g for 10 min enabling aliquots of the plasma
234 supernatant to be pipetted into labelled Eppendorfs and stored at -80°C for later quantification.

235 At the same time as blood was collected for the determination of GLP-1₇₋₃₆, a sample was also
236 obtained from the same puncture site to establish blood glucose concentrations. Each of these blood
237 samples (0.02 mL) was drawn into a sodium heparinised capillary tube and transferred into an
238 Eppendorf containing 1 mL haemolysis solution (EKF Diagnostics). Samples were shaken to
239 encourage haemolysis and then immediately placed on ice.

240 **6.2.8 Blood analysis**

241 Glucagon-like peptide-1 (GLP-1₇₋₃₆) was quantified by electrochemiluminescence, utilising a
242 human hormone multiplex assay (Sector Imager 2400, MesoScale Discovery, Rockville, MD,
243 USA). To reduce inter-assay variation, samples from each boy were analysed on the same assay
244 plate. The coefficient of variation (CV) was established as 5.5%. The blood glucose samples were
245 quantified by the glucose oxidase method using an automated glucose analyser (Biosen C line, EKF
246 Diagnostics).

247 **Statistical analysis**

248 Twenty one boys were included in the statistical analysis, as data for one of the boys was excluded
249 due to his EI being different in the requested period of replication prior to the first and second trials.
250 One more boy was unable to provide blood samples during the trials but all other data collected
251 from him was included in the analysis. IBM[®] SPSS (version 22.0, SPSS Inc., Chicago, Illinois) was
252 used for all analyses. Data was checked for normality using the using Shapiro-Wilk test and means
253 \pm SEM were calculated for all variables. Data from 20 boys was included for GLP-1₇₋₃₆ (pg/mL) and
254 blood glucose. To establish the effect of gaming, plasma GLP-1₇₋₃₆ (pg/mL) and blood glucose
255 (mmol/L) responses were calculated as time-averaged area under the curve (AUC) x 135 min for
256 both gaming trials. To establish the effect of the trials on subjective hunger, prospective food
257 consumption and fullness, VAS ratings (mm) were calculated as time-averaged AUC x 180 min.
258 Time-averaged AUC values for plasma GLP-1₇₋₃₆ and subjective appetite sensations, along with
259 gaming and test meal EI, gaming macronutrient EI (CHO, fat and protein), PA (METS), EE (MJ),
260 REI (MJ), time to eating onset during gaming (min) and the ingestion time of the test meal (min)
261 were analysed using paired t-tests. When significant differences occurred, Cohen's *d* effect size was
262 calculated and interpreted against the effect size categories of ≤ 0.20 = small effect, ~ 0.50 =

263 moderate effect, and ≥ 0.80 = large effect ⁽³⁵⁾. Significance was set at $p < 0.05$ for all analyses. To aid
264 in the interpretation of clinically meaningful statistical differences, time averaged AUC values x 90
265 min, determined from a between-variation study of fasted plasma GLP-1₇₋₃₆ ($4.8.1 \pm 0.1$ pg/ml) and
266 blood glucose (5.1 ± 0.0 mmol/l) in 8-11 y boys, were utilised (Allsop, S., Dodd-Reynolds, C.J.,
267 Green, B.P., et al., unpublished results).

268 **RESULTS**

269 **Population characteristics**

270 The 21 boys were of mean \pm SEM stature 1.45 ± 0.02 m, body mass 37.9 ± 1.6 kg, with a mean waist
271 circumference of 64.3 ± 1.7 cm and BMI of 18.1 ± 0.7 kg/m². According to UK age and sex-specific
272 BMI centiles ⁽³⁶⁾, the majority of the boys were classified as having a healthy body mass (77.3%),
273 9.1% were overweight and 13.6% as obese. Mean maturity offset was -0.3 ± 0.3 y, indicating that as a
274 group the boys were 3.6 months from peak height velocity and of similar maturation status. All
275 boys were identified as being unrestrained eaters with a mean \pm SEM dietary restraint score of $1.8 \pm$
276 0.13 categorised as being average for boys of this age (1.53 ± 0.06) ⁽²⁶⁾.

277 **Physical activity and energy expenditure**

278 All values for PA (METs), EE and REI are displayed in table 1. Active video gaming elicited light
279 PA (METs) and EE which was significantly greater than the sedentary levels produced by seated
280 gaming (all $p=0.000$, moderate effect size $d=0.7$).

281 **Energy intake and relative energy intake**

282 Table 2 indicates that boys consumed significantly more when seated video gaming, compared to
283 active video gaming (small effect, size $d=0.3$). As a percentage of total EI (MJ), the boys consumed
284 significantly more CHO (58.3 ± 16.7 %, $p=0.004$, small effect size $d=0.3$) and protein (6.8 ± 4.4 %, $p=0.022$, small effect size $d=0.1$) but less fat (36.4 ± 14.7 %, $p=0.004$, small effect size $d=0.3$) during
285 the active video gaming bout, than during seated video gaming (CHO 49.3 ± 12 %; protein 6.2 ± 2.9
286 %; fat 44.5 ± 11.3 %), as illustrated in Figure 2. No significant difference was found in the average
287 time to eating onset (min) and similar amounts of the test meal were consumed for which there was
288 no significant difference in ingestion time (min).

290 As shown in Table 2, following both the active and seated video gaming bouts the boys were in a
291 positive REI state. Relative energy intake was significantly greater due to seated video gaming than
292 active video gaming ($p=0.000$, small effect size $d=0.4$). When accounting for the test meal, REI was

293 significantly greater at the end of the seated video gaming compared to active video gaming
294 ($p=0.000$, moderate effect size $d=0.6$).

295 **Plasma GLP-1₇₋₃₆ and blood glucose**

296 No significant differences were detected in baseline plasma GLP-1₇₋₃₆ ($p=0.199$) or blood glucose
297 ($p=0.676$) between active and seated video gaming. There were no significant differences in time-
298 averaged AUC x 135 min for plasma GLP-1₇₋₃₆ (Figure 3a) between active and seated video gaming
299 ($p=0.413$). Time-averaged AUC x 135 min blood glucose was significantly greater during active
300 video gaming ($p=0.037$, small effect, size $d=0.3$), in comparison to seated video gaming as
301 illustrated in Figure 3b.

302 **Subjective appetite**

303 There were no significant differences in baseline appetite values for hunger ($p=0.917$), prospective
304 food consumption ($p=0.204$) and fullness ($p=0.315$) between seated and active video gaming. No
305 significant differences were detected in time-averaged AUC x180 min for hunger ($p=0.884$),
306 prospective food consumption ($p=0.570$) or fullness ($p=0.733$) between the active and seated video
307 gaming trials.

308 **DISCUSSION**

309 The present study was the first to rigorously explore the satiety-related signals, plasma GLP-1₇₋₃₆
310 and blood glucose in response to active and seated video gaming in 8-11 y boys. *Ad-libitum* gaming
311 EI and subjective appetite sensations were also measured during 90 min of gaming and in a post-
312 gaming test meal, to determine whether acute compensation occurred for gaming EI.

313 The main findings were that the *ad-libitum* gaming EI of 8-11 y boys was significantly greater
314 during seated than during active video gaming. Moreover, EI during both trials was a considerable
315 proportion of daily EAR for energy, for males aged 9 y, in the UK (7.70 MJ) ⁽³⁷⁾. Time-averaged
316 AUC blood glucose was significantly higher during the active video gaming trial (t=0 min to 135
317 min). Upon examination of the macronutrients revealed during the 90 min active video gaming
318 bout, the boys were found to have consumed a greater proportion of CHO during this trials, even
319 though overall EI was more when seated. The substantial EI during 90 min of both seated and active
320 gaming did not result in less EI in the test meal, 1 h later. In addition, the active gaming EI of the
321 boys was not offset by the estimated energy expended. Consequently, at the end of both 90 min
322 gaming trials, REI was positive which then increased following consumption of the post-gaming
323 test meal, although this was significantly higher for seated video gaming. Plasma GLP-1₇₋₃₆ was

324 raised during both trials but did not differ significantly. Subjective sensations of hunger, prospective
325 food consumption and fullness were also no different between trials. However, the subjective
326 responses appeared to reflect the increase in GLP-1₇₋₃₆ during 90 min of game play and decrease at
327 cessation of both trials. These latter findings are consistent with those found between seated gaming
328 and resting in male adolescents ⁽¹⁴⁾ and between seated video gaming and television viewing in 9-13
329 y boys ⁽³⁸⁾.

330 In the present study, the total EI of the boys during 90 min of seated video gaming was 2.64 MJ,
331 whilst in the active video gaming bout, EI was significantly lower (1.63 MJ). Per hour, the values
332 obtained for seated video gaming (1.75 MJ·h⁻¹) are identical to those found previously in 8-11 y
333 boys (1.75 MJ·h⁻¹) ⁽¹¹⁾ although higher than those observed in 8-12 y children (1.57 MJ·h⁻¹) ⁽³⁹⁾. For
334 active video gaming, EI was less per hour (1.08 MJ·h⁻¹) than previously established both in 8-11 y
335 boys (1.41 MJ·h⁻¹) ⁽²¹⁾ and 8-12 y children (1.60 MJ·h⁻¹) ⁽³⁹⁾. Differences in EI between studies could
336 be due to variations in study methodologies, since the current study was situated in the laboratory as
337 opposed to the after-school setting employed in our previous work ⁽²¹⁾. The more familiar and
338 relaxed setting of the school might have induced greater EI ⁽²¹⁾. In addition, Mellecker and
339 McManus (2008) ⁽³⁹⁾ utilised a seated video gaming device attached to a treadmill, instead of a
340 genuine active video gaming console ⁽³⁹⁾. The mode of active video gaming was less realistic and so
341 may have been less stimulating and challenging and this could have induced greater EI than
342 observed presently. In the present study however, the lower active video gaming EI may have been
343 due to the movement required for game play which could have made it more difficult to eat, than
344 when playing the seated video game. In addition, the longer delay in time to eating onset during
345 active video gaming, although not significant may help to explain the lower EI. In our previous
346 work with 8-11 y males however, a significant delay was observed in time to eating onset (min)
347 during active video gaming, yet EI was not significantly lower than when seated ⁽¹¹⁾.

348 The EI of the boys during 90 min of active video gaming although significantly less than for seated
349 video gaming, when compared to daily EAR for energy ⁽³⁷⁾, was 34% and 21%, respectively of
350 requirements. When accounting for the estimated EE during the bouts, the REI at cessation of
351 seated and active video gaming was 2.26 MJ and 0.99 MJ, respectively. Allowing for the post
352 gaming test meal, at the end of both trials, REI increased to 3.34 MJ for seated and 2.06 MJ for
353 active video gaming. Although the boys did not compensate for the extra EI during active video
354 gaming by down-regulating their EI at the test meal, the resultant energy surplus was 1.28 MJ lower
355 than when the boys were seated. Nonetheless, the REI for both active and seated video gaming may

356 be clinically meaningful in relation to weight status, as a reduction of only 0.46 to 0.69 MJ per day
357 may be all that is required to reduce the energy gap and decrease children's body mass ⁽⁴⁰⁾.

358 As previously reported with television viewing, it is possible, that both seated and active video
359 gaming could have a distractive effect which can lead to over-consumption of energy without an
360 increase in appetite sensations ⁽⁴¹⁾. Such an effect is thought to disrupt the habituation to food cues
361 from the sensory, neuronal and digestive systems ⁽⁴¹⁾ and as a consequence satiety signals appear to
362 be ignored thus causing over-compensation in EI for the energy expended ^(14; 38; 42). In relation to
363 active and seated video gaming, this disruption, coupled with an environment in which highly
364 palatable foods are available *ad-libitum*, could have activated the brain's reward centre. As a result,
365 satiety peptides might have been down-regulated and instead an augmented release of hormones
366 associated with pleasure (dopamine, endocannabinoids and opiates) may have been increased. This
367 response is related to hedonic systems and sustains the drive to eat ⁽²³⁾.

368 The only paediatric study ⁽¹⁶⁾ thus far to have investigated compensation due to active video gaming
369 EE, also did not establish any difference in EI in a post gaming meal, when compared with 1 h of
370 resting and seated video gaming. In contrast to the present study, at the end of the active video
371 gaming trial, the boys were in negative REI although this was compensated for 24 h later by an
372 increase in EI ⁽¹⁶⁾. However, food was offered *ad-libitum* in a post gaming test meal only and not
373 both during and following each trial, as in the present study. From the current study, it is not known
374 whether the boys compensated for the extra EI during both gaming trials at a later time, either by a
375 down-regulation in EI or an increase in EE. If no compensation occurred, the REI established for
376 both seated and active video gaming could contribute to a positive energy balance state and if it is a
377 frequent behaviour, could lead to obesity and so pose a risk to child health. Further research should
378 consider the effects of active video gaming on subjective and objective appetite over a longer time
379 period, to establish if compensation for the extra EI occurs more than 1 h post in 8-11 y boys.

380 The higher time-averaged AUC concentrations of blood glucose and lower EI of the boys during
381 active video gaming most likely occurred from the greater proportion of CHO consumed or the
382 greater EE. However, greater concentrations of blood glucose are also in accordance with the
383 'glucostatic theory'. The responses of glucose and GLP-1₇₋₃₆ which increased over the course of the
384 90 min of active gaming may have enhanced satiety and caused this lower EI. Nonetheless, during
385 both active and seated video gaming, EI was substantial and so an hedonic response should not be
386 dismissed as this may have superseded the homeostatic signal of GLP-1₇₋₃₆ to stop eating, during

387 active video gaming^(24; 43). Future active gaming studies should therefore consider the measurement
388 of insulin and hedonic food intake by VAS.

389 To the authors' knowledge, the present study initiated the investigation of the acute effects of active
390 and seated video gaming on glucose and GLP-1₇₋₃₆, in 8-11 y boys. In addition, fingertip capillary
391 blood sampling was utilised to quantify GLP-1₇₋₃₆ as this was only recently established as a
392 comparable option to the antecubital-venous method,⁽³⁴⁾. Considering the age of the present study
393 population (8-11 y), this alternative blood sampling technique provided a more suitable method for
394 the collection of blood and thus assessment of GLP-1₇₋₃₆ and glucose.

395 The measurement of hormonal appetite during active video gaming in children is not without
396 limitations. Due to the short half-life of GLP-1₇₋₃₆, to ensure its preservation, the study was
397 conducted in the laboratory rather than in a school setting as in our previous work^(21; 44). The levels
398 of glucose which were significantly greater during active video gaming than when the boys were
399 seated might also have stemmed from the low intensity exercise. As such, further research could
400 include the measurement of lactate to assess the demands of active video gaming⁽⁴⁵⁾. The present
401 sample was limited to the study of boys only, due to physiological aspects primarily related to the
402 different growth and maturation rates to girls during 8-11 y⁽⁴⁶⁾. For this reason the authors believed
403 it inappropriate to include girls, as to do this would have meant subdividing by gender and thus
404 reducing methodological rigour. Future paediatric active video gaming research in relation to EI and
405 appetite would therefore, benefit from work with girls.

406 To conclude, 90 min of active video gaming decreased EI, yet similar to seated gaming, REI was
407 positive following 90 min of active video gaming and this was not compensated for in the post
408 gaming test meal. Instead, the lack of compensation in the post-gaming test meal resulted in an
409 increase in REI, which although reduced by 1.28 MJ due to active video gaming, is a clinically
410 meaningful amount in terms of body mass⁽⁴⁰⁾. Active video gaming and food and drink
411 consumption should not therefore, be simultaneous behaviours in children, as this type of eating
412 behaviour could counteract the health benefits of the higher EE, which may then lead to an increase
413 in body mass.

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- 526

527 **TABLES**

528 **Table 1**

529 **Table 1.** Serving size, total energy and macronutrient values of food and drink items served during the gaming bouts.

Food or drink	Serving size	Energy (MJ)	Carbohydrate (g)	Fat (g)	Protein (g)
Apples (“Royal Gala” raw, sliced and cored)	130 g	0.26	15.60	0.13	0.52
Walker’s [®] ready salted crisps	50 g	1.10	25.75	15.95	3.05
Semi-skimmed milk	250 mL	0.52	12.00	4.50	9.00
“Jucee” apple and blackcurrant squash (no added sugar)	250 mL (1:5 dilution)	0.03	2.50	0.00	1.30

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539 **Table 2**

540 **Table 2.** Mean \pm SEM gaming EI (MJ), time to eating onset during gaming (min) PA METS, energy expenditure (EE) (MJ), gaming relative energy
541 intake (MJ), test meal EI (MJ), total relative energy intake and ingestion time of test meal (min) for all boys (n=21) for each gaming trial.

	Seated video gaming		Active video gaming		<i>p</i> value
	Mean	SEM	Mean	SEM	
Gaming EI (MJ)	2.65	0.32	1.63	0.26	<0.001*
Time to eating onset during gaming (min)	7.50	2.32	9.11	2.41	0.811
PA METS	1.22	0.04	1.99	0.11	<0.001*
EE (MJ)	0.39	0.01	0.64	0.03	<0.001*
Gaming relative energy intake (MJ)	2.26	0.32	0.99	0.26	<0.001*
Test meal EI (MJ)	1.08	0.12	1.07	0.10	0.859
Total relative energy intake (MJ)	3.34	0.35	2.06	0.30	<0.001*
Ingestion time of test meal (min)	11.02	4.53	8.48	3.33	0.051

542 *Indicates a significant difference between the active and seated video gaming trials.